Conservative treatment of acute kidney injury

By

Mohammed Wahba

Lect. Of internal medicine

Dialysis unit

MUH

Acute kidney injury (AKI)

- **Acute kidney injury (AKI) or acute renal failure (ARF), as it was previously termed is defined as an abrupt or rapid decline in renal filtration function. This condition is usually marked by a rise in serum creatinine concentration or by azotemia. However, immediately after a kidney injury, BUN or creatinine levels may be normal, and the only sign of a kidney injury may be decreased urine production.**

Categories of AKI

Categories of AKI

AKI may be classified into 3 general categories, as follows:

- Prerenal
- Intrinsic
- Postrenal

Oliguric and nonoliguric patients with AKI

- Classifying AKI as oliguric or nonoliguric on the basis of daily urine excretion has prognostic value. Oliguria is defined as a daily urine volume of less than 400 mL and has a worse prognosis, except in prerenal injury.
- Anuria is defined as a urine output of less than 100 mL/day and, if abrupt in onset, suggests bilateral obstruction or catastrophic injury to both kidneys.
- Stratification of renal injury along these lines helps in diagnosis and decision-making (eg, timing of dialysis) and can be an important criterion for patient response to therapy.

RIFLE Classification System for Acute Kidney Injury

Stage	GFR** Criteria	Urine Output Criteria	Probability
Risk	SCreat† increased × 1.5 *or* GFR decreased >25%	UO‡ < 0.5 mL/kg/h × 6 h	High sensitivity (Risk >Injury >Failure)
Injury	SCreat increased × 2 *or* GFR decreased >50%	UO < 0.5 mL/kg/h × 12 h	High sensitivity (Risk >Injury >Failure)
Failure	SCreat increased × 3 *or* GFR decreased 75% *or* SCreat ≥4 mg/dL; acute rise ≥0.5 mg/dL	UO < 0.3 mL/kg/h × 24 h (oliguria) *or* anuria × 12 h	High sensitivity (Risk >Injury >Failure)
Loss	Persistent acute renal failure:	complete loss of kidney function >4 wk	High specificity
ESKD*	Complete loss of kidney function	>3 months	High specificity

Acute Kidney Injury Network Classification/Staging System for AKI

Stage	Serum Creatinine Criteria	Urine Output Criteria
1	Increase of ≥0.3 mg/dL (≥26.4 μmol/L) or 1.5- to 2-fold increase from baseline	< 0.5 mL/kg/h for >6 h
2	>2-fold to 3-fold increase from baseline	< 0.5 mL/kg/h for >12 h
3	>3-fold increase from baseline, or increase of ≥ 4.0 mg/dL (≥35.4 μmol/L) with an acute increase of at least 0.5 mg/dL (44 μmol/L)	< 0.3 mL/kg/h for 24 h or anuria for 12 h

Treatment

Approach Considerations

- Measures to correct underlying causes of acute kidney injury (AKI) should begin at the earliest indication of renal dysfunction. Serum creatinine does not rise to abnormal levels until a large proportion of the renal mass is damaged, because the relationship between the glomerular filtration rate (GFR) and the serum creatinine level is not linear, especially early in disease. Indeed, the rise of serum creatinine may not be evident before 50% of the GFR is lost.

- It cannot be overstated that the current treatment for AKI is mainly supportive in nature; no therapeutic modalities to date have shown efficacy in treating the condition. Therapeutic agents (eg, dopamine, nesiritide, fenoldopam, mannitol) are not indicated in the management of AKI and may be harmful for the patient.

Treatment

Maintenance of volume homeostasis and correction of biochemical abnormalities remain the primary goals of treatment and may include the following measures:

- Correction of fluid overload with furosemide
- Correction of severe acidosis with bicarbonate administration, which can be important as a bridge to dialysis
- Correction of hyperkalemia
- Correction of hematologic abnormalities (eg, anemia, uremic platelet dysfunction) with measures such as transfusions and administration of desmopressin or estrogens

Vasodilators

- The rationale for vasodilator therapy in AKI is that improved renal perfusion may reduce renal damage. Strong evidence in support of this approach is lacking, however.
- A meta-analysis of 16 randomized studies concluded that the vasodilator fenoldopam reduces the need for renal replacement therapy and lowers the mortality rate in patients with AKI. However, larger trials need to be conducted before the use of fenoldopam can be recommended.
- Dopamine in small doses (eg, 1-5 mcg/kg/min) causes selective dilatation of the renal vasculature, enhancing renal perfusion. Dopamine also reduces sodium absorption; this enhances urine flow, which helps to prevent tubular cast obstruction. However, most clinical studies have failed to establish this beneficial role of low-dose dopamine infusion, and one study demonstrated that low-dose dopamine may worsen renal perfusion in patients with AKI

Dietary Modification

Dietary changes are an important facet of AKI treatment. Restriction of salt and fluid becomes crucial in the management of oliguric renal failure, wherein the kidneys do not adequately excrete either toxins or fluids.

Because potassium and phosphorus are not excreted optimally in patients with AKI, blood levels of these electrolytes tend to be high. Restriction of these elements in the diet may be necessary, with guidance from frequent measurements. In the polyuric phase of AKI, potassium and phosphorus may be depleted, so that patients may require dietary supplementation and IV replacement.

Calculation of the nitrogen balance can be challenging, especially in the presence of volume contraction, hypercatabolic states, GI bleeding, and diarrheal disease. Critically ill patients should receive at least 1 g/kg/day protein but should avoid hyperalimentation, which can lead to an elevated blood urea nitrogen (BUN) level and water loss resulting in hypernatremia.

Prevention of Contrast-Induced Nephropathy

- **Saline**

  In patients undergoing imaging studies with contrast, prophylactic administration of IV fluid has been shown to decrease the incidence of contrast nephropathy. Although controversy exists regarding the ideal fluid, normal saline and isotonic $NaHCO_3$ have proved to be effective. A normal saline solution of 1 mL/kg/h administered 12 hours before the procedure and then 12 hours after the procedure is recommended for most patients.

Prevention of Contrast-Induced Nephropathy

- *$NaHCO_3$*

  In patients who are at high risk for volume overload—in particular, those with chronic heart failure who have a left ventricular ejection fraction of less than 40%—isotonic $NaHCO_3$ solution should be administered before and after the procedure. It can be prepared by mixing 3 ampules of $NaHCO_3$ in a liter of 5% dextrose in water (D5W) and can be given at a rate of 3 mL/kg/h for 1 hour prior to the procedure, with the rate decreased to 1 mL/kg/h during the procedure and for 6 hours afterward.

Prevention of Contrast-Induced Nephropathy

- **N -acetylcysteine**

  Another prophylactic agent, used with varying success, is oral *N* -acetylcysteine at a dosage of 1200 mg every 12 hours. This is administered to high-risk patients the day before a contrast study is performed and is continued the day of the procedure. Diuretics, nonsteroidal anti-inflammatory drugs (NSAIDs), and possibly angiotensin-converting enzyme (ACE) inhibitors should be withheld near the time of the procedure.

  Any protective effect of *N* -acetylcysteine would appear to be limited to patients receiving radiocontrast. A meta-analysis of patients undergoing major surgery found no evidence that *N* -acetylcysteine used perioperatively can alter mortality or renal outcomes when radiocontrast is not used. Even among patients receiving radiocontrast, *N* -acetylcysteine appears to provide only borderline benefit.

- ***Other agents***

  Similarly, a review of randomized, controlled trials of other measures used to protect renal function perioperatively (eg, the administration of dopamine, diuretics, calcium-channel blockers, NSAIDs, ACE inhibitors, or hydration fluids) found no reliable evidence that these interventions are effective.

Long-Term Monitoring

Renal recovery in most cases is not complete, with the kidneys remaining vulnerable to the nephrotoxic effects of all therapeutic agents. Therefore, agents with nephrotoxic potential are best avoided.

Renal recovery is usually observed within the first 2 weeks, and many nephrologists tend to diagnose patients with end-stage (ie, irreversible) renal failure 6-8 weeks after the onset of AKI. It is always better to check these patients periodically, because some patients may regain renal function much later.

THANKS